

Some Aspects of Tropical Diseases as Seen at Home

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PATIENTS who return from the tropics may present a great variety of clinical syndromes. They may present themselves as a pyrexia of unknown origin, requiring special investigations for such conditions as malaria or visceral leishmaniasis, or with the dysenteric syndrome, requiring sigmoidoscopy and search for *entamoeba histolytica* in scrapings from the bowel. There may be undue loss of weight when sprue may enter the picture, or there may be a typical distribution of phrenic nerve pain produced by a liver abscess. Then there are the great varieties of helminthic infestations. The patient with ankylostomiasis may be profoundly anæmic, or he may have abdominal symptoms closely resembling those of duodenal ulcer. Cysticercosis must always be borne in mind in those who develop epilepsy; occasionally this condition may be caused by other infestations, such as cerebral schistosomiasis, especially in *schistosoma japonicum* infestations in people from the Far East. And so it is that tropical diseases can produce diverse symptoms and signs, from fever to sore tongue, abdominal tumours due to splenomegaly, general wasting, abdominal symptoms chiefly related to the large bowel, hæmaturia, and, more rarely, evidence of filarial infestation, such as lymph scrotum. Recognition of this wide variety of clinical evidence is important, for tropical infections can often be specifically diagnosed and specifically treated.

It is not possible in this paper to consider more than a very few points in relation to tropical diseases as seen at home, and so I propose to deal with the commonest condition, that is, malaria. This disease alone may present most varied clinical conditions, such as recurrent pyrexia, jaundice due to bilious remittent fever, or blackwater fever, coma, or mental symptoms due to cerebral involvement, or chronic ill health. In order to properly understand malaria as seen in the non-immune European, there are certainly fundamental facts first to be considered, for these are the basis of rational treatment with the various specific drugs, and especially paludrine.

Malaria infection is acquired under natural conditions in one way only, namely, by the bite of an infective female anopheline mosquito, i.e., the mosquito must have sporozoites in its salivary glands. Can naturally-acquired malaria occur at home? It is possible, for the local species of anopheles, *anopheles maculipennis* is an efficient vector, but the odds are against it, for the mosquito is cold-blooded, and development of the malaria parasite in the mosquito is very sensitive to the atmospheric temperature. However, mosquitoes lead a microclimatic life in relation to their immediate environment, which may by chance allow the full development of the parasite, and so transmission can occur, but it can only be sporadic. The disease

may be transmitted accidentally by inoculation if the needle is contaminated by the blood of a person harbouring the parasite. This probably accounts for the great majority of cases of home-made malaria. Blood transfusions from an infected donor may convey the disease; stored blood is safe if it is more than seven days old. Pregnant women who have a history of recurrent malaria should receive treatment with an anti-malaria drug, such as mepacrine or paludrine, for two weeks before and after delivery. This will prevent the accidental infection of the child during the process of birth (foetal infection is accidentally due to injury to the placenta during delivery: malaria cannot be truly congenital). It will also prevent the outbreak of malarial fever in the mother early in the puerperium, which is otherwise very likely to occur.

Transmission of malaria is a biological phenomenon directly related to the bio-nomics of the vector species of mosquito. After the female mosquito has been fertilised, she must have a blood meal before ovarian development can occur. If the species of anopheles has a desire only for human blood, then it is likely to be an efficient vector of malaria, e.g., anopheles minimus in Assam feeds almost entirely on human blood, which is one reason why that country is hyperendemic for malaria.

Some important points must now be considered concerning the behaviour of the malaria parasite when it gains entrance to the human body. When sporozoites are injected into an individual, the blood remains infective for a very short time only, possibly a few hours. It then remains sterile for seven to eight days until ring forms occur. This "negative blood phase" is important, for it is believed that the parasite during this time is lodged in the cells of the reticuloendothelial system. These exoerythrocytic forms have been demonstrated in avian malaria (*plasmodium gallinaceum*). It is on these forms of the parasite, or "cryptozoites," that paludrine has a particularly lethal action. Paludrine is also powerfully schizontocidal, and it also acts on gametocytes in such a way that it prevents their development in the mosquito beyond the oöyst stage. There are many strains of the various species of malaria parasite, e.g., the strains of *plasmodium vivax* in Africa is different from the strains in India, and the important point is this, that immunity in malaria is strain specific, except in the case of *plasmodium ovale*, which has only one strain. These different strains explain the different reports from various therapeutic measures in different areas. It is not possible to make general deductions about malaria from the study of the disease in one area only.

The drugs used in the treatment of malaria are quinine, mepacrine, pamaquin, and, more recently, paludrine. Quinine still maintains its place as an anti-malaria drug. It is schizontocidal, and in addition has an independent anti-pyretic action, which makes the patient more comfortable early in the disease. It should be given in an acid solution; pills should be avoided, as they are apt to pass through the intestine unchanged. It is certainly the drug of choice for parenteral administration. The drug is rapidly excreted and the dose is gr. 10 thrice daily. If required to be given intravenously, the dose is gr. 5 in 10 c.c. water given very slowly, and it is useful to precede it by pituitrin to prevent fall in blood-pressure. Quinine should

never be given if there is any suggestion of blackwater fever. The toxic effects are deafness, tinnitus, and erythematous skin rashes. Mepacrine is a synthetic yellow dye which is given in tablet form. Its action is similar to that of quinine. The usual dose used is 0.1 gr. thrice daily. It stains the skin and urine yellow, but not the conjunctivæ. The chief value of pamaquine is in B.T. malaria to prevent relapses. It may cause abdominal pain and should be given after a meal. Occasionally hæmoglobinuria may occur. The usual dose is 0.01 gr. twice daily. The action of paludrine has already been considered. It has no toxic effects even in large doses. Myelocytes may appear in the peripheral blood during treatment. The best dosage is not yet decided, but 100 mg. twice daily should be adequate.

It will often have to be decided, in those who return from the tropics, how long can malaria infection persist. As a general rule benign tertian malaria will be spontaneously eradicated after three years, and malignant tertian malaria after eighteen months. The quartan parasite can probably persist for many years, probably up to seven years. Patients who have pyrexia after these periods should be carefully scrutinised for infections other than malaria, especially kala-azar or amoebiasis.

In conclusion, it may be said that tropical diseases, as seen at home, may present a great variety of clinical evidence. Malaria has been specially considered as it is the commonest disease imported from abroad, and because it is important to recognise the significance of the disease and give proper treatment and advice to the patient.

REVIEW

NOTES ON INFANT FEEDING. By G. B. Fleming, B.A., M.D., F.R.C.P. (Lond.), F.R.F.P.S., and Stanley Graham, M.D., F.R.C.P.(Ed.), F.R.F.P.S. Pp. 66. 3s.

In this small book of some sixty pages, compiled primarily for the use of medical students of Glasgow University, and now in its third edition, the authors have condensed all the essential principles of infant feeding.

The difficulties in the maintenance of breast feeding are rightly emphasised: the suggested management is practical and sound physiology.

In the chapter on artificial feeding the caloric requirements of the normal infant for basal metabolism, growth, loss in excreta, and muscular exertion are examined in some detail, and, based on these considerations, the authors present a method of calculating the requirements of under-nourished infants, allowing for the changes in these four processes in the various degrees of malnutrition. While this may at first tax the memory and mathematics of the student, once he has become familiar with the method he can prescribe with confidence for any infant, whatever the age, weight, or nutritional state. While the practitioner accustomed to prescribe in ounces of milk per pound body weight will no doubt continue to do so, it is salutary when feeding problems arise to reconsider the principles of caloric requirements as discussed here.

There are short chapters on the premature infant, failure to thrive, and the management of cases of gastroenteritis, and many useful facts on development are included in an appendix.

This book succeeds in its object of giving the undergraduate a brief, readable, but solid foundation from which to build up the wider knowledge he will acquire in practice. J. B. T. L.